



## Standardized mortality ratios in early- and adult onset for three disorders

Lammers Vernal, Ditte; Grøntved, Simon; Ernst Nielsen, René; Briciet Lauritsen, Marlene

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therefore shortened by 1.4 years; excess LYs were 9.7 years in 1995–1999 (5.8/3.8 years due to natural/external causes) and 8.3 years in 2010–2015 (6.6/1.7 years due to natural/external causes). When looking at specific mental disorders, the life expectancy gap was reduced for mood disorders (0.8 years), neurotic disorders (1.7 years), and personality disorders (0.9 years); remained similar for schizophrenia spectrum disorder and substance use disorders; and increased for organic disorders (1.1 years).

**Discussion:** Mortality rates for people experiencing mental disorders decreased from 1995 to 2015. However, for natural causes of death, those with mental disorders did not reflect the benefits seen in the general population. Consequently, life lost due to natural causes increased. Overall, life expectancy increased an additional 1.4 years for those with mental disorders compared with the general population, thus reducing the gap. Nevertheless, for some disorders e.g. schizophrenia spectrum disorder and substance use disorders, life expectancy gap did not change. These findings support the hypothesis that service improvements have reduced mortality due to suicide and accidents, but similar benefits are not apparent in natural causes of death, which suggests that interventions related to promoting a healthier lifestyle and optimizing the general medical care of those with mental disorders warrants added investment.

### O7.5. STANDARDIZED MORTALITY RATIOS IN EARLY- AND ADULT ONSET FOR THREE DISORDERS

Ditte Lammers Vernal<sup>\*1</sup>, Simon Grøntved<sup>1</sup>, René Ernst Nielsen<sup>1</sup>, Marlene Briciet Lauritsen<sup>1</sup>

<sup>1</sup>Aalborg University Hospital,

**Background:** Severe mental illness is associated with a reduced life expectancy as compared to the general population. Furthermore, in many studies, early-onset of a severe mental disorder has been associated with a worse prognosis compared to adult-onset. In the current study early-onset groups of patients with schizophrenia, bipolar disorder or depression are compared on standardized mortality ratios (SMRs) to adult-onset.

**Methods:** The study is a register-based cohort study of the Danish population with the use of the Danish Register of Causes of Death, The Danish Civil Registration System, Danish Patient Registry and the Psychiatric Research Register. The cohort consisted of all patients diagnosed with schizophrenia, bipolar disorder or depression who were alive and living in Denmark during part or all of the study period from January 1, 2000 to the end of 2014. Patient diagnosis was available from the Danish Patient Registry as well as the Danish Psychiatric Research Registry from 1965 until end of study. Patients were followed from inclusion within the study period until age of 40 years old where patients were censored, death or end of study, whichever came first. If patients had received more than one of the included diagnoses, we grouped patients according to a hierarchy with schizophrenia being ranked highest, then bipolar disorder and lowest depression.

Early-onset (EO) was defined as a diagnosis prior to 18 years and for each psychiatric disorder and adult-onset (AO) as diagnosis between 18–40 years of age.

The primary outcome was SMR for all-cause mortality. As secondary outcomes, we looked at SMRs for unnatural death between each diagnostic group and the general background population. Unnatural death was defined as death by suicide, violence or accident. Early-onset patients (EO) were compared to adult-onset (AO) patients on all outcomes defined above.

**Results:** The total population consisted of 4,661,271 persons (51.1% males), of which 27,753 were diagnosed with schizophrenia (62.1% males), 13,925 with bipolar disorder (40.5% males) and 107,963 with unipolar depression (either single episode or recurrent) (34.5% males).

Compared to the general population, SMRs were > 1 in all groups, for both early-onset and adult-onset. Patients with early-onset schizophrenia had a SMR of 7.4 (95% CI: 4.7–11.7) as compared to adult-onset schizophrenia which had a SMR of 8.5 (95% CI: 7.6–9.6). Early-onset bipolar disorder

patients had a SMR of 5.9 (95% CI: 2.2–15.7) compared to the patients with adult-onset bipolar disorder 6.0 (95% CI: 4.7–7.6). In patients with unipolar depression with early-onset a SMR of 2.7 (95% CI: 1.8–4.0) was shown as compared to a SMR of 3.7 (95% CI: 3.3–4.1) in patients with adult-onset unipolar depression.

When investigating SMRs for unnatural death results were as follows: Schizophrenia EO = 11.0 (95% CI: 6.7–17.9), schizophrenia AO = 16.9 (95% CI: 15.2–18.6); bipolar EO = 9.5 (95% CI: 3.6–25.3), bipolar AO = 17.6 (95% CI: 14.4–21.5) and depression EO = 9.1 (95% CI: 6.3–13.3), depression AO = 9.6 (95% CI: 8.7–10.6).

**Discussion:** SMRs were > 1 for all three disorders for all-cause mortality as well as unnatural deaths.

Overall, we did not show worse relative outcome between the investigated groups of early-onset versus adult-onset patients within the three patients groups. Numerically, adult-onset appeared to have higher SMRs than early-onset in schizophrenia and depression, and in all three groups for unnatural death, but the differences did not reach statistical significance.

### O7.6. NEIGHBOURHOODS MATTER TOO: ASSOCIATION BETWEEN NEIGHBOURHOOD SOCIO ECONOMIC DISADVANTAGE AND TYPE 2 DIABETES COMORBIDITY IN SERIOUS MENTAL ILLNESS

Abstract not included.

## O8. Oral Sessions: Environmental Risk

### O8.1. ASSOCIATION BETWEEN GENETIC AND ENVIRONMENTAL RISK FOR SCHIZOPHRENIA DURING UPBRINGING: FINDINGS FROM A LONGITUDINAL COHORT STUDY

Joanne Newbury<sup>\*1</sup>, Louise Arseneault<sup>1</sup>, Avshalom Caspi<sup>2</sup>, Terrie Moffitt<sup>2</sup>, Candice Odgers<sup>3</sup>, Dan Belsky<sup>4</sup>, Tim Matthews<sup>1</sup>, Helen Fisher<sup>5</sup>

<sup>1</sup>King's College London; <sup>2</sup>King's College London, Duke University;

<sup>3</sup>Duke University; <sup>4</sup>Columbia University; <sup>5</sup>Institute of Psychiatry, Psychology & Neuroscience, King's College London

**Background:** Associations of environmental exposures such as urban upbringing, deprivation and crime victimization with psychosis are well-established. An enduring question, however, is whether associations reflect a causal process. Emerging evidence using polygenic risk scores (PRS) suggests reverse causation, with adults at higher genetic risk for schizophrenia being more likely to live in crowded and deprived areas. This process could occur due to the downward mobility of individuals at higher genetic risk for schizophrenia into more disadvantaged environments. However, the handful of studies on this topic to date have typically focused on environmental conditions during adulthood and have typically examined only macro-level environmental features (e.g., urbanicity).

**Methods:** We examined associations between two measures of genetic risk (PRS for schizophrenia and family psychiatric history) with multiple features of children's family-, neighborhood-, and wider- environments during upbringing. Data were from the Environmental Risk (E-Risk) Longitudinal Twin Study, a nationally-representative cohort of 2,232 British twins born in 1994–1995 and followed to age 18 (with 93% retention). Environmental risk factors were measured from early childhood to late adolescence, and included urbanicity, air pollution, neighborhood deprivation, neighborhood crime, neighborhood disorder, social cohesion, family poverty, residential mobility, and crime victimization. At age 18, participants were privately interviewed about psychotic experiences (e.g., hallucinations and delusions) occurring since age 12.